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ORIGINAL MEMOIRS.

LUDWIG'S ANGINA.

AN ANATOMICAL, CLINICAL AND STATISTICAL STUDY.*

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LUDWIG in 1836, described a condition which he considered a morbid entity, and which since that time has been designated, more or less universally, as Angina Ludovici. Various attempts have been made to establish it upon a definite pathological basis, but the results of none of these can be said to have been generally accepted. That it is an infection there can be no doubt, but the character of the infection if it has a special character, has never been decided. That it is more rapidly fatal than similar infections occurring in other parts of the neck than the submaxillary region, is amply proved by the recorded cases, but why or how it acquires so dangerous a character, has never been clearly demonstrated. It is well known that certain cases assume a grave aspect and threaten or take the life of the patient in twelve to twenty-four hours, while others begin and continue as a comparatively mild affection for days and then suddenly assume an alarming character. That its general course and symp-

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toms are typical and essentially constant is convincingly shown by the numerous cases that have been reported. Yet many have been and some are still being reported, which should not be designated by this term. The following case led the writer to make a study of the subject.

W. W., male, age 32 years, machinist, admitted to drunk ward of the Philadelphia Hospital, August 10, 1903. On admission the temperature was 98, pulse 110, respiration 30. Has been drinking for about a month. Is nervous and has marked tremors of the hands and tongue. He cannot eat or sleep. The heart is rapid but the sounds are good, and there are no murmurs. He has no hallucinations, and is well nourished. He complains of a small, painful swelling under the right side of the lower jaw which has been there for about a week. On August 13 he was transferred to the surgical ward, in the service of Dr. A. C. Wood, to whom the writer is indebted for the privilege of reporting the case. Temperature 98.2, pulse 78, respiration 22. The swelling is increasing in size. The pain keeps him awake at night and prevents him from taking his nourishment. Incision made in the submaxillary region, the index finger being introduced its entire length without evacuating any pus. August 14th swelling is increasing rapidly, is hard, non-fluctuating, and involves the whole under side of the jaw. Speech is difficult, and he is having such difficulty in swallowing that he is being fed with a spoon. Temperature 101, pulse 100, respiration 26. General condition otherwise good. August 15, at 8 P.M., he was cyanotic and respiration was very difficult. Tongue swollen. Oxygen inhalations given for a time with some relief. Then he became rapidly worse. Pulse intermittent, rapid and weak. At 10 P.M. tracheotomy was performed by the resident in charge, Dr. Speese. Incision below the cricoid cartilage. Profuse bleeding from the veins in front of the trachea. Surrounding tissues very œdematous. From the time the trachea was opened the patient's condition rapidly became worse, and although he breathed through the tube, he could not be kept alive by artificial respiration, which was continued for about fifteen or twenty minutes.

Autopsy—Pathological Diagnosis.—Œdema of the glottis; unilateral interstitial nephritis; hemorrhagic infiltration of intestinal mucosa. The tissues about the glottis and epiglottis are

intensely swollen. This swelling is so extensive about the glottis that only a chink, about 2 mm. in breadth and 6 mm. in length of the glottis remains.

The writer regards this case as a typical Ludwig's Angina. The following case was reported, September 4, 1905, before the North West Medical Society of Philadelphia, as a "Gun Shot Wound of the Lower Jaw, followed by Submaxillary Cellulitis, simulating Ludwig's Angina." Since then a study of the literature has shown other cases, reported as Ludwig's Anginas, which were essentially of the same type. The writer considers that this grade of infection in this region has every dangerous characteristic, indeed somewhat exaggerated, of a typical Ludwig's Angina. The reader is referred to the later discussion on etiology and pathology, for the writer's reasons for including it here as an example of this disease.

J. W., colored, age 31 years, admitted to the Philadelphia Hospital, August 9, 1905, in the service of Dr. A. C. Wood, with whose permission the writer reports the case. His general health and strength were excellent.

On August 8th, in a quarrel, the patient was shot twice by a revolver in the hands of a companion, who stood in front of him, and about five or six feet away.

There are three wounds of the face, one of which is a well-rounded and perforating wound of the cheek, about three-eighths inch in diameter, just to the right of the symphysis. A second wound with irregular edges is situated on the right cheek, about one inch in front of the lobule of the ear. A fragment of a bullet was removed from this wound. The third perforation, which was so insignificant and covered by stubby beard that it was not discovered for a few days, is shaped like the first, and is situated on the left cheek about two and a half inches posterior to the angle of the mouth. The probe enters this wound for about two inches, when it strikes what at first was thought to be the ramus of the jaw. A skiagraph later showed a bullet lodged in the tissues in about the situation of this opening. At first the patient did not complain of this wound, and it was then thought that

the two on the right side were produced by the two bullets. It seems evident now that they were due to a single bullet which entered near the symphysis, struck the jaw, splintered it, and was divided, one fragment glancing off and producing the wound in the right cheek near the lobule of the ear. Both wounds of the right cheek met within the mouth at the injured portion of the jaw.

The tissues of the interior of the mouth, internal and external to the jaw, are intensely swollen, particularly internal to the jaw in the floor of the mouth. The tongue almost fills the mouth and interferes with normal respiration. Speech and deglutition are disturbed. There are four teeth missing in the lower jaw in the right molar region. The patient says the teeth were not missing before the shooting. In the space corresponding to the missing teeth the alveolar border of the lower jaw is splintered, the loose fragments being removed with forceps. There is a complete fracture of the lower jaw on the right side about an inch anterior to the ascending ramus. The wounds were all washed out and packed with gauze, dressings applied, and a cardboard cup was fitted to the jaw and held by a Barton's bandage.

On the following day the patient's condition became alarming on account of the difficulty in breathing. The face was more swollen, particularly in the submaxillary region. The tongue and the floor of the mouth were more swollen than on the preceding day, and the tissues in the floor of the mouth were more brawny to the feel. The patient was etherized and the two wounds on the right side of the face were enlarged into the mouth. All loose fragments of bone and soft tissue were removed and the wounded tissues irrigated with boric solution. An incision about two and a half inches long was made parallel with the lower jaw and about midway between the hyoid bone and jaw. This wound was deepened until the finger was close to the mucous membrane of the floor of the mouth in the region of the damaged portion of the lower jaw. Irrigation and dressing as before. Temperature 101.3°, pulse 128.

On the following day, August 11th, his condition had improved slightly, but the swelling and temperature were about the same. Respiration, deglutition and speech were still disturbed. On examining the region of the injury to the jaw, the wound in the floor of the mouth was found covered with gan-

grenous sloughing tissue, and the odor was very foul. The wounds in the lip and cheek leading away from this region were discharging foul pus. A mouth gag was introduced on the opposite side of the mouth and the tongue held away from the infected area, thus exposing it. After clearing away all shreds of gangrenous tissue and irrigating with boric solution, the infected surface was cauterized with pure carbolic acid, which was neutralized at once by applications of alcohol. The patient was placed in charge of a special nurse, who cleansed the infected region every half hour with peroxide of hydrogen and boric solution.

On the following day a marked improvement was noticeable. The swelling was evidently decreasing, the patient could talk, swallow and breathe better, and said that he felt much better. In the few succeeding days the temperature fell to normal. The discharge was still copious and offensive. The septic condition soon subsided and the case resolved itself into one of healing wounds of the face and neck and fracture of the lower jaw, which later united.

History.—Parker, in 1879, published an interesting historical review of this condition as recorded before the appearance of Ludwig's paper, with particular reference to the cases occurring in England. He gives some details of a case referred to by Auretius which seem to have been those of a Ludwig's Angina. He called the condition, "cynanche." Paulus Aegineta spoke of a somewhat similar condition, which he called "paracynanche." Many of the older authors, both Greek and Arab, including Hippocrates, Galen, Celsus, Aurelianus, Rhases and others, had described the disease. Dr. Fothergill gave an account of "Putrid Sore Throat" (1739–1746), which appeared to have some of the characteristics of Ludwig's Angina. He also gave an historical review of what is believed to have been the same disease. Dr. Kirkland in 1786, Dr. Wells in 1809, and others reported cases of this type.

It remained, however, for Ludwig to present the first accurate description of this dangerous condition, which he called "gangrenous induration of the neck." Cameror, in

the following year was the first to apply to it the name, Ludwig's Angina. Following the appearance of Ludwig's paper considerable interest was manifested and an increasing number of cases were reported. Probably, greater interest has never been shown during any one period, than that which was bestowed on it by the French Surgical Society, in 1892. Several successive meetings were devoted to it and many cases cited by those present. There was a marked difference of opinion manifested, which culminated in a division of the members into two parties, one being led by Nelaton, the other by Delorme. Nelaton took the stand that Ludwig's Angina should not be recognized as a separate disease, and was instrumental in having resolutions to this effect passed by the society. At the following meeting Delorme caused this action to be reconsidered and Ludwig's Angina to be given its proper place in surgical pathology. In the following year Leterrier published a thesis in which he reported 27 cases collected from the literature and communicated four new ones, three of Delorme's and one of his own. The chief object of his paper appeared to be to support the position of his teacher, Delorme, who contended that Ludwig's Angina was primarily a sublingual phlegmon.

In the same year, 1893, Poulsen published the results of a study of 530 abscesses of the neck collected from hospital statistics. In 1886, he had presented a paper in which he reported his observations on a series of lime injections under the deep fascia of the neck, to prove the existence of communicating channels of loose connective tissue between the various adjacent interfascial spaces. In his second paper he attempted to show that infections tended to follow these channels and to invade the various spaces, successively. His explanation of the progress of the infectious process in a Ludwig's Angina will be taken up later in the discussion of the etiology and pathology of this disease.

In 1895, Semon's paper appeared, in which he maintained that acute septic inflammations of the throat and Ludwig's Angina were pathologically identical, and should be

included together as one group of diseases, thus eliminating Ludwig's Angina as a separate disease. Since that time nothing new has been offered on this subject so far as the writer can learn.

Etiology and Pathology.—Although fairly authentic cases were recorded before, practically, nothing was presented in the literature to establish the cause and nature of this condition, until Ludwig's paper appeared. Since that time many cases have been reported and much has been written, which is of value in clearing up the obscurities surrounding Ludwig's Angina. Yet its etiology and pathology still remain obscure. In the writer's opinion, one of the basic causes of confusion lies in the obscurity associated with the cause of death, in connection with which, the chief question is as to whether it results from septic intoxication or from invasion of the air passages. Probably, both conditions are always present to some degree, in typical cases; but the relative importance of each has never been established. If septic intoxication is the essential cause of death, then the especially high mortality of this condition is to be explained by the presence of a rare and especially virulent infection. If invasion of the respiratory tract is the dangerous feature, peculiar to this condition, then the mortality is to be explained by extension of the phlegmonous inflammation to the larynx and in some cases to the lungs. Upon the solution of this question depends, in the writer's opinion, the explanation of the etiology and pathology of Ludwig's Angina.

Ludwig suggested that it was epidemic in its nature, that it was allied to erysipelas and that it was a true morbid entity. Every one of these suggestions has been supported and combatted vigorously by many different authorities and it may fairly be said that they remain unsettled up to the present time. Tissier, Roser, and Chabri, for example, agree with Ludwig, as to its being a morbid entity. On the other hand, Boehler, who collected and studied 35 cases, refused to accept this view and tended to suppress the name of Lud-

wig's Angina. v. Thadden gave to it the name of "submaxillary bubo," while Chantemesse considered it a true erysipelas of the larynx. Roser believed that the disease began in the submaxillary salivary gland. This theory has not been borne out by the post-mortem evidence which has been accumulated. It will not be profitable to discuss here more than a few of the theories which have been offered as to the etiology and pathology of this condition, and it is particularly, to the later authorities that the writer will confine his attention.

As already indicated the investigations have followed two distinct channels; the bacteriological, which attempt to prove that a particular type of infection and therefore a septic intoxication is responsible for the condition; and the anatomical, which try to show that the condition is due to the particular location of the infection and its peculiar opportunities for dangerous extension.

Influence of Septic Intoxication:—Definite and positive convictions on the relative importance of septic intoxication, can not be reached without difficulty. In studying this phase of the subject, first importance should be given to the bacteriological findings. A search of the literature has shown the following cases in which different bacteria were found and reported.

Delorme, staphylococcus in one case and streptococcus in another; Leterrier, undetermined bacillus in one; Macaigne and Vanverts, pneumococcus predominating, with streptococcus and staphylococcus in one; Lockwood, streptococcus, staphylococcus and bacillus of malignant oedema in one of his own cases and in another, streptococcus, cocci and diplococci. In Gibson's case he also found the streptococcus; Biedert and Robertson, streptococcus in one; Gasser, streptococcus and bacillus coli communis in one; Ross, streptococcus and staphylococcus in one; Davis, streptococcus alone in two cases, and streptococcus and staphylococcus in a third; Ombredanne and Keim, streptococcus and staphylococcus in one; Humphrey, pneumococcus alone in one; Duplay, staphy-

lococcus in one; Chantemesse and Widal, streptococcus in one; Magnal, streptococcus in one.

It will thus be seen that of the 18 cases, the streptococcus was found alone in 6 cases; the streptococcus associated with other organisms in 8; the staphylococcus alone in 2; the pneumococcus alone in one; and an undetermined bacillus in one.

The fact that stands out most prominently in this group of cases is that the streptococcus was present in almost all, either alone or associated with other bacteria. That it may be present in some cases in which the investigation has failed to show it, may be inferred from the fact that Lockwood, by different methods, found the streptococcus in Gibson's case, although Cameron reported that he could find "no specific microörganisms in the tissues." In all the writer's collection of cases, the inflammation of the connective tissues has appeared to be of a severe type, and in a considerable number a gangrenous or fetid process has been present. The inference to be drawn from these facts is that a severe septic infection and a corresponding grade of septic intoxication has been encountered. Yet in many cases the constitutional symptoms have been only moderate or very mild. Even if they were severe in all, this would not show that they were the cause of the high mortality, since the same infections occurring in other parts of the body, giving as severe local and constitutional symptoms, do not produce the same death rate as does Ludwig's Angina. Since the existence of a special infection, capable of explaining the high mortality, has been searched for, carefully, by qualified investigators without success in a fairly large number of cases (probably many more than the writer has found record of), we may assume with some confidence, that none such is present. The clinical as well as the post mortem evidence, so far accumulated, is decidedly against the existence of such a cause; while the evidence in favor of ordinary severe types of infection, particularly, the streptococcus is very strong.

Ludwig, whose description of the clinical course, has remained the standard up to the present, said that in the

first four or five days, the constitutional symptoms were not severe, but became so later. From a study of 104 cases collected from the literature and his own two, the writer believes that this change in gravity of the constitutional symptoms, has a definite relation to the invasion of the mouth and pharynx by the phlegmonous process: and that the increase in severity is out of all proportion to the increased area infected, and the corresponding amount of toxins absorbed. This raises the question as to whether the constitutional symptoms are due entirely to septic intoxication, or whether they may not be due in part to interference with respiration. Davis says "whether these deaths are due to suffocation or heart failure caused partly by sepsis and partly by the impeded respiration is sometimes difficult to say." He also adds that "these sudden deaths occur usually in patients in which the epiglottis and larynx are affected and the dyspnoea marked." One would infer from this statement that Davis believes that these sudden deaths are the result of the affection of the epiglottis and larynx. The writer believes that practically all deaths in Ludwig's Angina are to be accounted for in the same way. Some develop pneumonia and pleurisy, while a few may die of septic intoxication. Engelman says that seventy-five per cent. of children dying of diphtheria have broncho pneumonia. Diphtheria is a severe infection of essentially the same parts of the throat as are involved ultimately in these cases of Ludwig's Angina, and broncho pneumonia should be as likely to result in one as in the other. Septic intoxication, itself, probably, kills no more patients suffering from Ludwig's Angina, than do these same types of infection occurring in other parts of the body, as in the palm of the hand, the forearm or leg, or in other parts of the neck. "In Robertson and Biedert's case," Davis says "sudden death occurred after a tracheotomy had been performed, so that suffocation could not have been the cause." While it would be difficult to show that suffocation, actually, occurred in this case, the fact that the first symptom complained of was dyspnoea, and that six hours after the onset

it was so severe that tracheotomy became imperative, points to the fact that disturbance of the respiratory tract probably killed the patient. In this case as in most of the 14 which Semon reported, the phlegmonous process, evidently, began close to the larynx. In Semon's cases extension to the lungs or pleurae occurred in 5 out of the 6 fatal cases. Pneumonia developed in 3, in one on both sides, and in two double pleurisy was present. In two of the eight cases, which recovered, a double patchy pneumonia was noted. On the same point Davis says further: "In one of Ross' cases, likewise, sudden death resulted while the opening existing through the larynx was sufficient to preclude respiratory obstruction." In this case the focus from which the phlegmonous inflammation extended was, evidently, the necrotic wisdom tooth, and from this focus pus and gas escaped on prying away the tooth. With the beginning of the process only about two inches away from the larynx and within the mouth close to the pharynx, it is more than likely that oedema of the larynx developed early. On the fourth day after operation, two patches of impaired resonance were made out, one in each lung. It would seem to be evident, therefore, that in both these cases, the invasion of the respiratory tract and not septic intoxication, caused the death of the patients. Why these cases in which the clinical evidence of oedema of the glottis, *i.e.*, the intense dyspnoea, is so pronounced as to demand immediate tracheotomy do not recover when this operation permits an apparently free passage of air to and from the lungs the writer is not prepared to explain. That the deaths in these are, indirectly or directly, the result of the invasion of the respiratory tract, larynx alone or larynx and lungs, the writer believes. One of his own cases breathed through the tube after the tracheotomy had been performed, but could not be kept alive by artificial respiration. In one of Baker's cases, tracheotomy was done soon after his admission to the hospital, but the pulse stopped during the operation and the patient died. The autopsy showed oedema of the glottis (see autopsy cases). In one

of Tissier's cases, tracheotomy was performed for intense dyspnoea on the day of his admission to the hospital, the third day of the disease. Notable relief followed the operation, but the patient died the same night. In Weiss' case, a tracheotomy was done on the first day of the disease. It was necessary to continue artificial respiration for a half hour to revive him. He recovered. Fenwick's case required a tracheotomy, 4 hours after the beginning of the disease. Great relief followed the operation, but the patient died three hours later. In Gibson's case, swelling began in the neck below the lower jaw, at noon of one day. On the following day the swelling was enormous, extending to the chest and zygoma. The floor of the mouth was considerably thickened, and there was slight dyspnoea. He was admitted to the hospital about 1 P.M. At 3 P.M. of the same day, he became intensely dyspnoeic and tracheotomy was performed immediately, followed by artificial respiration. He recovered and the respiration became normal. On the next day at 11 P.M. there was dyspnoea and considerable cyanosis of the face and lips. He gradually became comatose and died at 3.15 P.M. The autopsy showed oedema glottidis (see autopsy cases). There can be little room for doubt that in all these cases the essential cause of death was the invasion of the respiratory tract, larynx alone or larynx and lungs. Septic intoxication, probably, played only a secondary part in bringing about the fatal result.

It is well known that the partial obstruction of the pharynx from faucial and pharyngeal adenoid growths, will impair the general health of a child by interfering with the normal respiration. Much greater interference coming on suddenly in Ludwig's Angina, from pushing the tongue upwards and backwards and crowding the mouth and pharynx should produce a more serious deleterious effect upon the general condition, the signs of which will be added to and confused with those of the septic intoxication which is already present. When we take into consideration the fact that there was oedema of the glottis in, practically, every fatal case in

the writer's group of cases, in which the larynx was afterwards exposed at autopsy, it becomes evident that the interference with respiration is greater than is generally supposed. Dyspnoea was noted in nearly all the fatal cases, and in the opinion of the writer it is the invasion of the larynx and lungs, not the septic intoxication, which is the peculiarly dangerous feature of Ludwig's Angina. It is sufficient to explain the high mortality, septic intoxication is not.

While in most of the cases it is difficult or impossible to differentiate between the parts played by these two factors, in a few it is shown clearly that all the alarming symptoms characteristic of a Ludwig's Angina may develop in the absence of severe constitutional symptoms, as in the following. Where temperature alone is given it should be borne in mind that this was the only symptom mentioned in the report of the case, from which one could infer the degree of the constitutional disturbance; and where it is not given here it was not mentioned in the report, and any statement implying the degree of constitutional disturbance or absence of it was extracted and employed in these brief summaries. In one case reported by Huguet and DeBovis, there was an extensive submaxillary swelling, "enormous" sublingual swelling, dysphagia, dyspnoea and a considerable quantity of fetid pus; yet the temperature never went above 39°C (102°F). In one of Parker's cases, the usual severe symptoms were present except dyspnoea, which may have borne some relation to the presence of a discharging sinus in the floor of the mouth. This may have checked the progress of the inflammation towards the larynx. The general health was not impaired. In another of Parker's cases, the general health was reported to be good. Leube's case, which underwent resolution, had a normal temperature. In Trump's case and in three of Davis' cases, the temperature was only 101°F. In Margerison's, the temperature was 100.8°F, pulse 104, and in Humphrey's it was never above 100°F. Leterrier reported that in his case the general condition was good, the temperature 37.4°C (99.3°F) and that the patient

would have taken food if he could swallow. All these cases recovered. Michel's patient was admitted to the hospital on the 5th day of the disease, when he had an enormous submaxillary swelling. On the day preceding admission asphyxia was threatened. He died 4 hours after admission. The temperature was given at 39°C (102°F). One of Schwartz's cases, on the day of admission to the hospital, insisted on going out again to attend to some business, which he was permitted to do. He returned later in the day and died of syncope that night. In Gibson's case, the submaxillary swelling began at noon of one day. On the following day at 1 P.M., when he was admitted to the hospital, the swelling was enormous. A little later the dyspnoea became intense. Tracheotomy was performed and artificial respiration carried out with relief to the patient. At 3 P.M. of the same day he died in coma and dyspnoea. Yet the temperature on admission, 2 hours before death, was only 97.8°. In Fenwick's case, the swelling began in the morning. Two hours later the face was almost unrecognizable. In 4 hours he was cyanosed and could hardly breathe, and in 7 hours he was dead. Yet the temperature was norml, the pulse 140. It would seem, therefore, that in some cases essentially all the symptoms of a Ludwig's Angina may be present, and those of septic intoxication be very moderate or practically absent. Indeed, in only a comparatively small number of the cases collected by the writer, was high temperature referred to, and in the great majority the presence of severe constitutional disturbance could only be inferred from the general gravity of the case. Inspection of the atopsy cases, given later, will confirm this statement.

Influence of the local condition.—While definite results have never been obtained from bacteriological investigations, beyond the fact that the streptococcus is present in nearly all the cases, pure or mixed with other organisms; the study of the local inflammatory conditions have yielded more satisfactory results: The observations of Poulsen, Delorme, Semon and more recently Davis, in the writer's opinion, have

been the most valuable of recent years. These writers seemed to consider the infection from a distinctly local standpoint, and to regard the larynx as the essentially vulnerable point of attack.

Poulsen says that the deep cervical fascia in the submaxillary region is dense and resistant, and that the submaxillary salivary and lymphatic glands are enclosed in a fascial space. This submaxillary fossa communicates by means of loose cellular tissue and blood vessels with the deep retromaxillary fossa, so that a cellulitis beginning in one of these spaces readily extended to the other through this communicating passage. He explains the dangerous symptoms of dyspnœa and dysphagia in Ludwig's Angina, by an extension of the inflammation through the wall of the pharynx to the pharynx and larynx from the retromaxillary fossa. He contended that those cases beginning with a preliminary angina gave secondary involvement of the lymphatic glands in the retromaxillary fossa about the bifurcation of the carotid artery, and that the resulting periglandular cellulitis then passed through the wall of the pharynx. When the phlegmonous process began in the submaxillary lymphatic glands, as from a carious tooth or ulcer in the tongue or floor of the mouth, the overlying strong fascia gave rise to great tension so that the inflammation, seeking the direction of least resistance, passed along the communication to the retromaxillary fossa, and thence through the wall of the pharynx to the pharynx and larynx. Poulsen's conclusions are not based upon strictly anatomical studies, but upon the results of his lime injections. When the lime was injected under the deep fascia in the submaxillary region, it first produced a swelling in this region which was soon followed by extension to the region of the large vessels of the neck, and almost simultaneously to the alveolo-lingual sulcus in the floor of the mouth. In no case did it work its way through the wall of the pharynx, the path by which Poulsen claimed that the inflammation reached the larynx. He obtained hospital statistics of 530 abscesses of the neck, of which 251 occurred in the submaxillary region. Of the 251, there was a swelling in the floor of the mouth or alveolo-lingual sulcus in 22. In 2 of the 22 there was a spontaneous opening in the floor of the mouth, in one at the orifice of Wharton's duct. As a rule the inflammation

subsided after incision in the submaxillary region, and only twice was the œdema so abundant that an incision in the mouth was necessary. Of the 251, 11, or 4 per cent., died. Poulsen considered that only three corresponded to the clinical picture of Ludwig's Angina, in which he attached especial significance to the non-fluctuating swelling in the submaxillary region, the lack of large pus foci, the intact skin, and the extension of the swelling to the floor of the mouth. He eliminated one of these because of the absence of an autopsy. The writer has included the other two in his collection of cases, and they will be found among the autopsy cases.

The two points in Poulsen's paper, to which the writer attaches greatest importance are: first, that Ludwig's Angina results from the extension of an infection of the neck to the larynx and pharynx; and secondly, that the cellulitis had its origin in extension from the lymphatic glands. He was far, however, from proving the path of extension. His most important evidence lay in the fact that in several cases, when the abscess was opened the finger of the surgeon could be passed down to the pharyngeal wall, the infection being traced in this way nearer to the pharynx and larynx than in any other direction. He attached considerable significance to the fact that in one case, not regarded by him as a Ludwig's Angina, during the making of an external incision into the abscess, there occurred a spontaneous opening into the throat. In no cases did he demonstrate an opening in the pharyngeal wall. Spontaneous openings have been reported rather frequently, generally in the mouth, some of them occurring near the base of the tongue or in the throat, and are readily explained in another way.

As the result of his clinical observations and experience Delorme concluded that Ludwig's Angina was nothing more than a sublingual phlegmon; although on account of its exact anatomical seat and constant symptoms, he was inclined to view it as a morbid entity and to retain the name of Ludwig's Angina. Leterrier in his thesis, already referred to, offered two arguments to support Delorme's theory. In the

first place it was found necessary in all their cases to cut through the mylo-hyoid muscle from the neck, and, therefore, into the sublingual tissues, before pus was reached. In the second place, according to Leterrier, the almost constant swelling in the floor of the mouth and the elevation of the tongue, could be due only to a sublingual phlegmon. He also added that when there was a spontaneous opening made by the pus, it was usually internal. He believed that if this theory was generally accepted and the external incision extended deeply enough, the mortality would be much diminished. All of their cases recovered. A number of writers, particularly in France, accepted Delorme's view and reported Ludwig's Angina as synonymous with sublingual phelgmon. Huguet and DeBovis, who collected and studied 49 cases, regarded them as sublingual phlegmons, but held that "these sublingual phlegmons can only be the result of diffusion of an inflammation developed more posteriorly in the region of the parotid or angle of the jaw." They believed that its anatomical seat was intramuscular, *i. e.*, that it was a basic glossitis. They could not admit that a purulent collection under the mucous membrane in the floor of the mouth would produce a hard, non-fluctuating swelling; and they added that some surgeons who have intervened by the mouth have not met with success or have had to plunge the bistoury to a considerable depth.

With reference to this point the writer has investigated his 104 collected cases with the following rather indefinite results. Nelaton made a sublingual puncture, only blood escaping. Later he made two external incisions, one a suprahyoid incision exposing a putrid focus, the other a submaxillary incision only infected serum escaping. Death resulted from syncope. No autopsy. Chauvel made a double sublingual incision and exposed a gangrenous focus above the mylo-hyoid muscle, extending to the upper border of the thyroid cartilage. (Extension to the thyroid cartilage implies that the focus was below the mylo-hyoid muscle also, and therefore in the neck.) Dubois found phlegmonous pus

by a sublingual incision. Haering made buccal scarifications but found no pus. Cuffe made a buccal incision toward the posterior part of the tongue but found only blood. Later the incision was repeated and pus was found. Holthouse made buccal scarifications but found no pus. Ross found no pus by a sublingual incision, but with an external incision located a large abscess. Ripault evacuated 2 or 3 cupfuls of pus by a buccal incision, and by a median external incision also found pus. There were sublingual and retromaxillary fluctuation in this case.

In most of the cases, however, it was the external incision which located the pus, and in only a few was the mylohyoid muscle said to be divided. The writer will show later that the sublingual phlegmon is the result of extension in the great majority of the cases, and that it is not the primary phlegmon as Delorme maintained. Leterrier explains the origin in the sublingual tissues by assuming that the infectious germs gaining entrance by a focus in the mouth as a carious tooth or an ulcer, are carried by the lymphatics to the cellular tissue about the sublingual gland. He says also that Richet has described a chain of lymphatic glands arranged in a horse shoe manner along the internal surface of the inferior maxillary bone, thus implying that if these glands existed, they would explain the frequency of cellulitis by periglandular extension.

Semon's paper, which appeared in 1895, is the most recent to attract wide attention. His conclusions are based upon clinical observations on 14 cases, which he saw in 20 years of special practice as a laryngologist. The main conclusion he reached was that "these acute septic inflammations of the throat and neck, described by a large variety of terms, such as acute oedema of the larynx, oedematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx, and Angina Ludovici, are pathologically identical. They merely represent different degrees of severity of one and the same septic process due to invasion of the throat and neck by various micro-organisms." He

adds that this can be finally proved only by a harmonious combination of clinical, pathological and bacteriological evidence. In every one of his cases, except the first he had tried to obtain a bacteriological investigation, but only in the last was this opportunity afforded, and then the evidence was purely negative. He called attention to the fact that Virchow could not exactly define the mutual relationship between erysipelatous and phlegmonous affections. Semon believes that the question of the primary localization and subsequent development depends, in all probability, upon accidental breaches of the protecting surface, through which the pathogenic microörganisms gain entrance to the tissues.

According to Semon, therefore, we are not concerned with any particular infection, so much as with a special type of inflammation, an acute septic phlegmonous process, which may be due to various microörganisms. Lockwood, who studied this condition from the bacteriological side, reached the conclusion that Ludwig's Angina is a mixed infection of the most complicated kind, and that several pathological conditions are included in this affection. He found that usually the streptococcus was present, though not always; and that this microörganism may be present alone or associated with other organisms, as the staphylococcus. From his study of the subject the writer prefers to accept Semon's view on this point. The complicated nature which Lockwood assigned to this infection, becomes simplified by the fact that whatever microörganism is found, the process is always the same, a rapidly spreading phlegmonous infiltration of the cellular tissues. This is the result usually produced by streptococcus infection, and it may be due to staphylococcus infection. Gasser quotes Queno as saying that any of the pyogenic organisms may be found in these cases. Other organisms may also produce it, as the bacillus of malignant oedema, which Lockwood found in one of his cases. We are not yet familiar with the exact results produced by the various bacteria, and Semon's statement seems sound that "it is absolutely impossible to draw at any point a definite line of demarcation be-

tween the purely local and the complicated, or between the oedematous and suppurative forms."

Semon maintains, however, that all acute septic phlegmonous inflammations of the throat and neck should be classified together, and that Ludwig's Angina as a separate disease should be eliminated. That they are all pathologically identical and that the throat, *i.e.*, the larynx, is the most vulnerable point in all, the writer believes. From the standpoint of prognosis and treatment, however, there is a very practical difference between those in which the phlegmonous process begins in the throat and those in which it begins in the neck, where the condition described by Ludwig had its origin. Many of the latter have shown a preliminary angina, it is true, but this usually disappeared later and did not form a part of the phlegmonous process beyond serving as the portal of entrance for the microörganisms. In most of Semon's cases and in one of the writer's collected cases, the acute septic process began in the throat and spread out from there. These in the writer's opinion, form a distinct group, and are laryngological; those which Ludwig described are distinctly surgical and in the majority of cases respond to surgical treatment. The following advice given by Semon, may be proper for the former but not for the latter. "Should there be anywhere distinct fluctuation or merely justifiable suspicion of such, of course you will incise upon such foci. Our promise for the future must depend on the fact that we have a bacterial infection, and that by the injection of an appropriate antitoxine we may be able to save the patient." Fluctuation or even a suspicion of it is practically never present. Prompt and suitable incision in the absence of any sign of fluctuation, has arrested the progress of many cases, probably, after oedema of the larynx had already set in. Antitoxines may be employed with advantage after incision and drainage have been provided, but not before.

No fact is more evident from a study of the literature, or is so generally conceded, than that the cellular tissue is

the essential seat of the inflammatory process, and that the surrounding structures become involved by contiguity. The literature also shows clearly, notwithstanding the claims of Delorme and his followers, that in the great majority of cases the cellulitis originates externally in the submaxillary region and not in the sublingual region, *i.e.*, in the mouth. Of the writer's 106 cases, in 61 the swelling was first noted in the submaxillary region of one side. In 16 it was bilateral and under the jaw when first seen by the physicians reporting them. In 2 there was a submental swelling which may have been a bilateral submaxillary involvement. In 13 others the swelling was described as involving the cheek and neck, face and neck, parotid region, etc., *i.e.*, it was in the beginning an external swelling. Of the 106, therefore, 92 began in the tissues of the neck external to the mouth and throat. In 8 cases the first swelling was sublingual, and from the description in 3 (Huguet and DeBovis 2, Holthouse 1) the writer considers it doubtful whether a sublingual or a submaxillary swelling first appeared. In two cases (Tordeus and Aldrich) it was described as a submaxillary and sublingual swelling. One case began in the throat as in Semon's cases, and is included here because it was considered by the writers reporting it as a Ludwig's Angina. The writer regards these facts as of much value in establishing the nature of the disease, and considers that they support what Ludwig claimed, that the cellulitis begins in the submaxillary region, at least, in the great majority of cases. Those which begin in the mouth can be easily accounted for, but there has been much dispute concerning the submaxillary origin and the term, idiopathic, has been employed in connection with them. Semon says "A little abrasion on the side of the neck exposed to the action of those pathogenic organisms may, of course, invade the body from the outside and may cause what has hitherto been called an Angina Ludovici. The original focus is purely accidental." One would infer from this that Semon considers that from such an abrasion, invasion occurs by direct continuity of tissue until the throat is involved.

Davis says: "When the teeth are the starting point the inflammation involves the periosteum of the lower jaw and thence invades all the surrounding tissues. While the point at which the infection starts localizes the disease at its commencement, it progressively spreads and invades all the tissues within its scope. No matter how it commences it spreads along the connective tissues by direct continuity. It is not transmitted by the lymphatics. The lymphatic glands do not become enlarged by infection carried to them by the lymph stream from the infectious focus, but they are involved in the connective tissue surrounding them." As already stated Leterrier considered that the infection was transmitted from some focus in the mouth to the cellular tissue about the sublingual gland setting up a cellulitis there. Roser believed that the infection was transmitted to the submaxillary salivary gland, and that the extension occurred to the surrounding cellular tissue.

That the primary focus in the great majority of cases is some insignificant lesion in the mouth, as a carious tooth, an herpetic or other ulcer, a tonsillitis, etc., has been generally admitted and so far as the writer can learn has never been denied. In many, however, no such focus was discovered. If the infection gains entrance to the tissues by such a focus in the mouth and the signs of inflammation first appear in the submaxillary region, external to the mouth and some distance from the original focus, the process can not be said to have extended by direct continuity of tissue. This applies with greater force to those in which no preliminary focus could be found, the typical, so-called, idiopathic cases. The writer's statistics on this point will be found later in connection with the clinical course of this condition.

There can be only one explanation for such a transference of infection, and that is by way of the lymphatic vessels to the glands in the submaxillary region. Most infections in this region are of glandular origin. Poulsen said that the great majority of his 251 submaxillary abscesses were cases of simple or localized adenitis, and he takes it for

granted that his cases of Ludwig's Angina began also in the lymphatic glands. v. Thadden considered it a lymphatic disease and gave to it the name "Submaxillary bubo." Localization of infection is the rule in any part of the body, and this is particularly true of those which lodge in lymphatic glands. Fulminating cases are rare. Typical Ludwig's Angina is rare and is also fulminating. It is easily conceivable that such an infection might be transferred from some slight focus in the mouth, where there is no retention, the discharge being free, to a submaxillary lymphatic gland where the infection is confined, and therefore more active, and from there on account of its increased activity invade the periglandular tissue so rapidly that its glandular origin is overlooked. In some cases the glandular origin was indicated by an early localized pain in the submaxillary fossa, which was soon followed by rapid swelling.

While the glandular origin was concealed by the rapid swelling in most of the writer's cases, this was not true of all. In one of Tissier's cases there was pain in the left submaxillary region on the first day. Swelling appeared on the following day. In one of Delorme's cases, the condition was first observed in the submaxillary region as "three glands," rapidly increasing in size. Bauer reported one in which the patient had similar attacks before. Ludwig's case in 4 days, had only reached the size of a hen's egg. One of Haering's when first seen was of the same size, Heyfelder's the size of a goose egg, and Timpe's of a five franc piece. Davis says of two of his cases, that one week before, the neck began to swell and later increased rapidly. In Blasburg's case there was an indolent swelling for 8 days and rapid swelling began on the 11th day. In the writer's case there had been a small lump for about a week before rapid swelling began. There can be little doubt of a glandular origin in these cases, and in the writer's opinion, they go far toward proving the glandular origin in the so-called idiopathic cases.

(To be continued.)